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Rheumatoid Hand Deformities: Pathophysiology and Treatment

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Rheumatoid disease, as it affects the hand, is a disease of the synovium lining the joints and sheaths of the tendon. The proliferating synovium destroys the articular surfaces of the joint, interferes with the gliding mechanism of the tendons and weakens the supporting ligaments of the joints. The degree and variety of deformities is multifold.

Treatment of the rheumatoid hand is aimed at conservation and restoration of hand function, as well as prevention of future deformities. Rheumatologists, physical therapists and hand surgeons carry out important functions in the well-planned, integrated regimen. Surgical treatment of the rheumatoid hand deformity may alleviate pain, lessen deformity and improve function in selected cases. It should be integrated in the general medical management of a patient.

Treatment of tendon ruptures includes tenorrhaphy, tendon grafting and arthrodesis in the case of mallet finger deformity. The wrist joint is improved by synovectomy and carpal tunnel release is accomplished by median nerve decompression. Metacarpal phalangeal joint deformities may be treated by synovectomy or silastic joint replacement when there is destruction of the articular joint surface, severe subluxation, or persistent painful motion.

AN ESTIMATED 3.6 million adults in the United States have rheumatoid arthritis according to a recent United States Public Health Service survey.[1] In about 75 percent of patients with rheumatoid arthritis involvement of the tendons and joints of the hand develops. Initial changes include synovial proliferation and swelling which may progress to joint destruction with severe loss of function. A fuller understanding of the pathological process of rheumatoid hand disease by primary physicians and improved surgical techniques by hand surgeons afford the opportunity for a team effort to alleviate pain, prevent or lessen deformity, and increase function in these crippled hands. This paper will discuss in detail rheumatoid involvement of the hand and wrist and the resultant deformities and loss of function. The indications for and techniques of surgical correction are correlated with the pathologic abnormalities.

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Pathophysiology

Rheumatoid arthritis is a disease primarily affecting the synovial membrane which lines the joints and tendon sheaths. Joint and tendon changes are secondary to the rheumatoid synovitis.

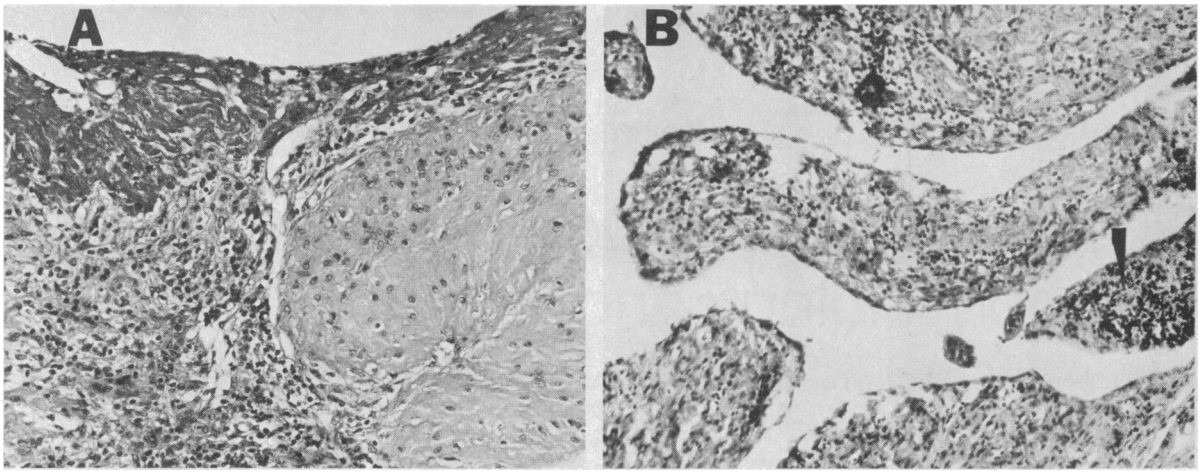

Figure 1.—Typical microscopic picture of rheumatoid arthritis illustrating (A) subsynovial fibrin of varying age with scant lymphocytic infiltrate and infiltrating granulation tissue (left pannus) exending over the articular cartilage, (B) Elongated and hyperplastic synovial villi with papillary hyperplasia and lymphocytic infiltrate and giant cell (arrow). (Photo courtesy Dr. John Maddox, Department of Pathology, Stanford University Medical Center.)

The normally thin synovial membrane proliferates to form a thick, villous edematous pannus. Microscopically, the synovitis is characterized by lymphocyte and plasma cell infiltration and increased vascularity with round cell infiltration, edema, and an increase in fibrous tissue in the perisynovial tissues (Figure 1).[2] The joint spaces become distended by overproduction of synovial fluid and proliferative synovium. The synovial reaction destroys the articular cartilage and attenuates the inflamed articular capsules and ligaments permitting the joints to be drawn into abnormal positions by muscle imbalance.[3] The diseased synovium surrounding the tendon invades the tendons with subsequent gradual dissolution of their normal fibers. There follows gradual stretching of the tendon, diminution in its strength and ultimately rupture. Dissolution of tendon may also be caused by interference with its blood supply by extrinsic pressure from the surrounding edematous tissues or bony prominences. The tenosynovitis disrupts the gliding mechanism of the tendon within the synovial sheath and limits tendon excursion.

Abnormalities at the Wrist

Early changes in the wrist include pain, swelling and limitation of motion. A proliferative synovial reaction may be visible on both the dorsal and palmar aspects of the wrist as a palpable, irregular, multilobular, boggy swelling. As the disease progresses, the restraining ligaments of the ulna gradually stretch and there follows a dorsal prominence of the ulnar styloid.[4] Several deformities are characteristic of advanced rheumatoid disease at the wrist. A flexion deformity of the wrist may result from the destruction of the radiocarpal joint and the relatively stronger action of the flexor muscles than the extensors of the wrist.[5] Grasping power of the fingers is reduced because of the check-rein effect of the stretched out long extensors of the fingers and the relatively slackened flexor tendons secondary to the flexed attitude of the wrist. The optimal function position of the wrist, about 30 degrees dorsiflexion, is lost.

Median Nerve Compression

The carpal tunnel syndrome of pain and paresthesia in the thumb, index, long and ring fingers, plus thenar atrophy, may be produced by rheumatoid involvement of the flexor retinaculum with median nerve compression. Similarly, proliferative synovium around the flexor tendons in the carpal tunnel may put pressure on the median nerve.[6] Other signs and symptoms include characteristic night pain, a positive Tinel sign (paresthesia produced by pressure or tapping over the median nerve area) and a reproduction of the symptoms by acute flexion of the wrist (Phalan test).[7] The diagnosis should be confirmed by neurophysiologic tests, such as electromyograms, nerve conduction velocities, and motor and sensory latency times across the wrist. The carpal tunnel syndrome may be one of the earliest manifestations of rheumatoid arthritis, hypothyroidism, gout, acromegaly or systemic lupus erythema-

tosus, and consequently the patient with this condition should be examined for these conditions and others.

Involvement of the Metacarpal Phalangeal Joints

Early manifestations of rheumatoid disease at the metacarpal phalangeal joints include pain, swelling, tenderness and limitation of motion. The joints become swollen periodically. In the later stages of the disease a flexion deformity of the metacarpal phalangeal joints with associated ulnar deviation of the finger is a constant finding in advanced rheumatoid disease. The lumbrical and interossei muscles are primarily responsible for flexion of the metacarpal phalangeal joints. Secondarily, these muscles act to extend the proximal and distal interphalangeal joints through their insertion into the extensor hood. Rheumatoid involvement of the intrinsic muscles produces spasm and subsequently fibrosis, thereby exaggerating the normal action of these muscles. The weakened metacarpal phalangeal joint is gradually drawn into acute flexion. Later, subluxation of the joint occurs. The ulnar deviation of the fingers is the end result of several factors: the relatively greater ulnar mobility of the metacarpal phalangeal joint, the pull of the lumbrical muscles, the obliquity in an ulnar direction of the metacarpal heads and the bow-string effect of the tendons which slip into the ulnar gutter adjacent to the joint.[8]

Involvement of the Fingers

Many varied abnormalities of the finger joints may be found in rheumatoid arthritis (Figure 2).[9,10] In fact, adjacent fingers in the same hand may exhibit a variety of altered pathological attitudes. The proximal interphalangeal joint may be hyperextended because of the intrinsic muscle contracture and the distal interphalangeal joint is flexed by the pull of the flexor profundus tendon which is tightened to its maximum by the hyperextended mid-joint. The profundus tendon thus flexes the distal phalanx in a check-rein fashion. This attitude of finger flexion of the metacarpal phalangeal and distal interphalangeal joints and hyperextension of the proximal interphalangeal joint is termed swan-neck deformity.

Mallet (baseball finger) deformity results from a rheumatoid tendon deformity which features attenuation of the insertion of the extensor tendon into the distal phalanx and a resultant incomplete extension of the distal phalanx. The Boutonniere

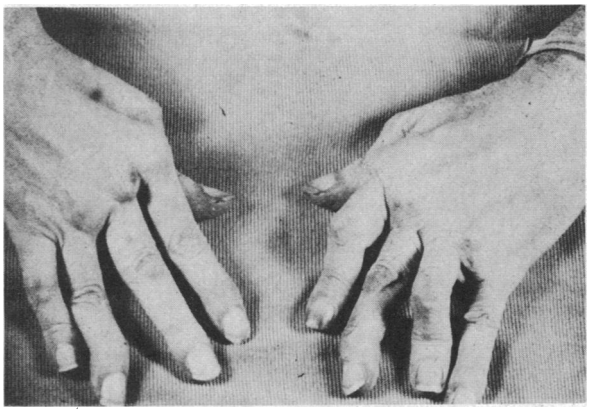

Figure 2.—Typical finger and thumb deformities in rheumatoid arthritis. Metacarpal phalangeal joints illustrate ulnar drift and subluxation, ring finger of right hand shows swan-neck deformity, index and ring finger of left hand illustrate boutonniere deformity, midfinger of left hand has mallet finger deformity, and both thumbs have typical deformity.

deformity is caused by attenuation and rupture of the central slip of the extensor tendon at the proximal interphalangeal joint with resultant incomplete extension at this joint and hyperextension at the distal interphalangeal joint. Trigger finger or stenosing tenosynovitis is produced by the encroachment of the proliferating synovium in the flexor tendon sheath. The tendon is eventually notched and sticks in flexion.

The thumb undergoes changes not unlike those seen in the other digits. The metacarpal phalangeal joint is drawn into flexion and the interphalangeal joint into hyperextension. Opposition of the thumb is further impaired by degeneration of its carpal metacarpal joint.

Tendon Ruptures

Tendon ruptures occasionally precede other manifestations of rheumatoid involvement of the hand. The most commonly affected tendons, sites of rupture and resultant deformities are the extensor pollicis longus at the Lister tubercle at the distal radius producing incomplete thumb extension; tendons of the extensor digitorum communis to the ring and little fingers at the wrist joint producing finger drop, and the extensor carpi ulnaris tendon at the same area producing inability to dorsiulnar flex the wrist.[11]

Treatment

Since rheumatoid arthritis is a systemic disease, the major effort in treatment is directed at control of the entire disease process. The current medical treatment consists of rest, large doses of aspirin

and general supportive measures such as heat, paraffin baths, gentle physical therapy and protective night splinting. Local and systemic steroids, gold salts, and a wide range of anti-inflammatory agents have application under the direction of a rheumatologic specialist. Only when medical management fails to arrest the progression of the disease in the hands is surgical therapy indicated (Table 1). All authors agree that surgical operation is more effective if undertaken before extensive damage to the joints and tendons has occurred. Prevention of deformity by early operation will give far better results than multiple surgical procedures on the almost hopelessly crippled hand resulting from long-standing disease and progressive deformity resulting from many years of neglect. Surgical operation is now frequently done even during active stages of the disease, and is directed toward preservation and restitution of hand function, as well as prevention of future problems. Operation in many cases should be preceded by medical treatment and physical therapy in order to gain the greatest range of motion of stiff joints before operation.[12] Each hand is treated separately.

Wrist Joint

If a pronounced flexion deformity of the wrist joint is present, the grasping and pinching power of the hand will be severely impaired. In some patients, the carpal bones will have almost completely disintegrated and the wrist joint dislocated. Arthrodesis of this joint may be necessary,[13] but frequently a synovectomy will suffice in the early stages of the disease.[4] Transposition of the extensor retinaculum from above to below the extensor tendons and resection of the ulnar head are adjunctive measures to prevent eventual tendon rupture.[5]

Median Nerve Compression

When signs of median nerve compression within the carpal tunnel are observed, the nerve must be decompressed. In the early stages of this syndrome, temporary relief may be achieved by the instillation of steroids beneath the ligament and night splinting. Definitive treatment consists of excision of a portion of the transverse carpal ligament through its entire length. Removal of the hypertrophic synovium from the carpal tunnel will further decompress the nerve and protects the tendons from further damage.

Finger Joints

Stenosing tenosynovitis or trigger finger is corrected by excision of the thickened tendon sheath with resultant freeing of the tendon. Baseball or mallet finger, the result of disruption of the lateral extensor bands as they insert at the distal phalanx, is best treated by primary arthrodesis, although tendon grafts or transfers to the distal extensor tendons may occasionally be attempted. This joint is arthrodesed in approximately 15 to 30 degrees of flexion.

Boutonniere deformity is extremely difficult to correct. In some instances a free tendon graft is used to span the midjoint. Worthwhile improvement can also be obtained by a tenotomy of the extensor tendon over the joint which relieves the strong pull of the lateral extensor bands and allows the distal joints to come into flexion.[9] Swan-neck deformity may be treated by intrinsic muscle release and transfer of tendon tension along the finger extensor.[10]

Tendons

A tenosynovial reaction surrounding the flexor and extensor tendons frequently forms a large spongy cystic mass (Figure 3). Early removal of

TABLE 1.—*Summary of Rheumatoid Hand Deformities*

Area Involved	*Deformity*	*Correction*
Synovium	Synovitis	Synovectomy
Wrist	Dorsal, volar synovitis	Synovectomy
	Dorsal subluxation ulna	Resection ulnar styloid
	Flexion deformity	Arthrodesis, arthroplasty
Median nerve	Carpal tunnel compression	Release carpal tunnel, synovectomy
Metacarpal phalangeal joints	Synovitis	Synovectomy
	Ulnar drift, subluxation	Silastic arthroplasty
Fingers	Swan neck	Tendon transfer
	Mallet finger	Arthrodesis
	Trigger finger	Decompression tendon sheath
	Boutonniere	Tendon repair

the diseased synovium is a requisite for the prevention of continued destruction of the gliding surface of the tendons. The involved synovium is carefully dissected from the tendons.[11] Frequently the tendons will be found to be attenuated and in some cases completely disrupted. These tendons are repaired, if at all possible, by end-to-end anastomosis. When the tendon is destroyed over a great length, or when tendon integrity is tenuous, it is simpler to restore action with the appropriate tendon transfer of a tendon having a similar and synergistic action.[14] (Example: the extensor indicis proprius tendon is transferred into the distal extensor pollicis longus tendon for thumb extensor rupture.)

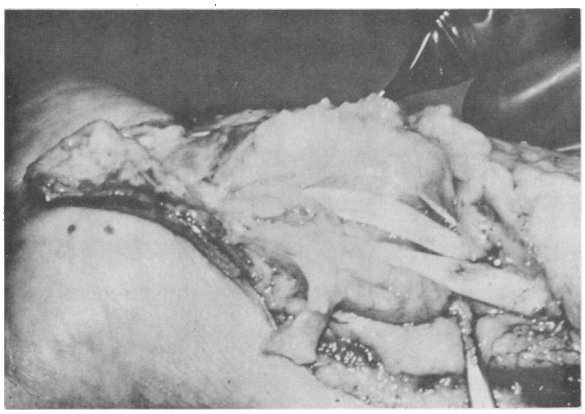

Figure 3.—Proliferative hypertrophic synovium surrounding and infiltrating extensor tendons at metacarpal phalangeal joints.

Metacarpal Phalangeal and Interphalangeal Joint

Synovectomy of the metacarpal phalangeal joints should be done early in the course of the disease to prevent destruction of the articular surfaces. At the time of synovectomy of the proximal joints, the extensor hood is repositioned to the radial side of the joint to correct the ulnar migration of these tendons.

Replacement of diseased joints by artificial ones has shown good results in many cases (Figures 4, 5). Swanson silastic prosthesic devices are the most commonly used, having been introduced in 1965.[15] The indications for the use of silastic prosthetic devices in the metacarpal phalangeal and interphalangeal joints include destruction of the articular joint surfaces with ulnar drift, severe

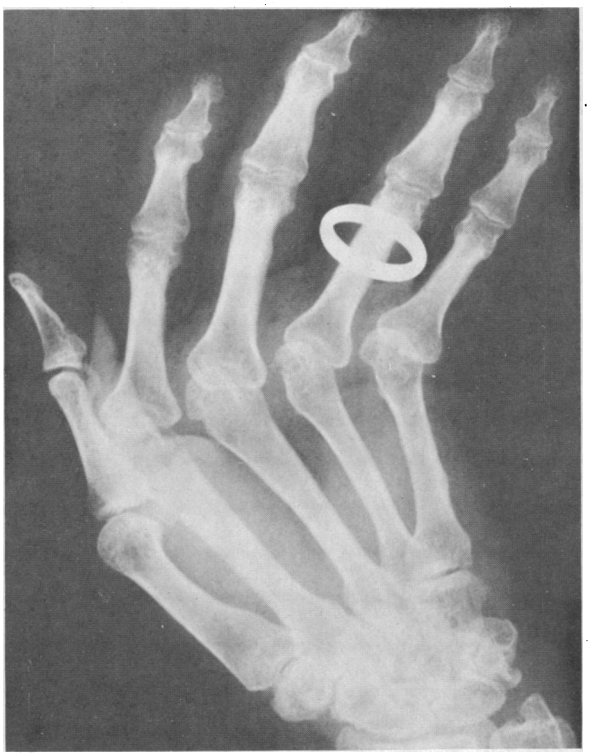

Figure 4.—Preoperative x-ray film of metacarpal phalangeal joints illustrating severe ulnar drift, joint destruction and subluxation.

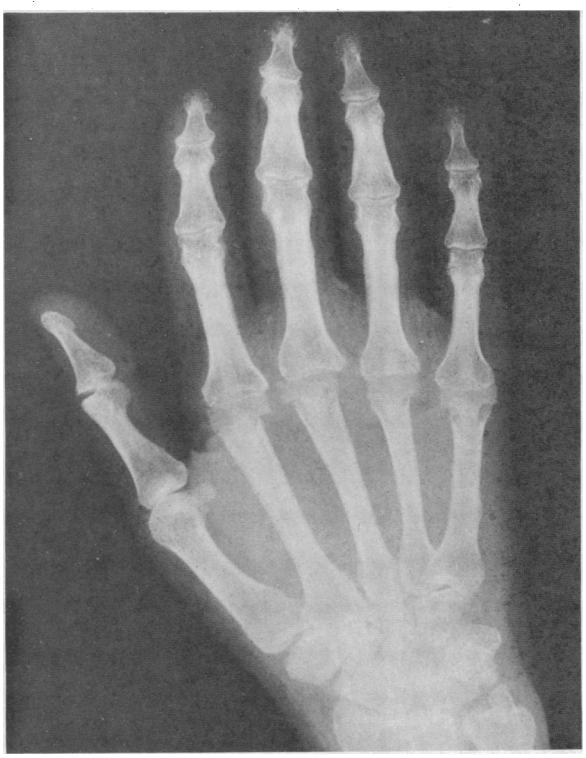

Figure 5.—Postoperative x-ray film of metacarpal phalangeal joints after silastic replacement arthroplasty. Note straight position of fingers with prosthetic joint in place and stems in bony medullary canals.

subluxation and persistent painful motion. Prosthetic joint replacement may be undertaken even in the face of active disease. There is no untoward tissue reaction to prosthetic material, nor has any case of rheumatoid deformity been activated by surgical operation. The greatest subjective relief is often a diminution of pain, as well as greater hand function through improved position and greater mobility.

When the proximal interphalangeal joints are severely deformed or subluxed, arthrodesis is the treatment of choice. This provides a stable pain-free joint. The loss of flexion is compensated by motion in the metacarpal phalangeal and distal joints. Arthrodesis is also indicated for instability of the distal joint of the thumb. These joints are fixed in 30 to 40 degrees of flexion for maximal functional use.

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Airway Impairment and Hypoxia

REGARDLESS of whether you have an anesthesiologist there or not . . . if you are looking at the airway becoming impaired—(and it really doesn't matter if you're talking about a patient in the trauma room or somebody with a tumor in his larynx or edema in the larynx that you're watching)—the thing that is going to kill that patient probably is hypoxia. The body has extremely small oxygen stores, and if you exclude whatever amount of oxygen is in the lungs, the total body oxygen store in the average adult is of the order of about a liter. The body is consuming about 200 cc a minute . . . That being so, while you are watching and thinking, the patient should have oxygen. And I would say, even in our own institution, that a very common error is to be called for some horrendous problem and the patient is not on oxygen. So the number one thing is that while you're thinking, give the patient oxygen.

—H. BARRIE FAIRLEY, MD, *San Francisco*

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